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VINYL CHLORIDE

An annotated bibliography

with emphasis on genotoxicity

and carcinogenicity

SEPTEMBER 1998

Ontario

Ministry
of the
Environment

ISBN 0-7778-7908-5

VINYL CHLORIDE

An annotated bibliography with emphasis on genotoxicity and carcinogenicity

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SEPTEMBER 1998

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PIBS 3698E

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Summary

The vinyl chloride monomer is the primary constituent in the manufacture of polyvinyl chloride (PVC) products. The vinyl chloride (VC) and polyvinyl chloride (PVC) industries in conjunction with their associated industries comprise a significant proportion in the industrial sector in many industrial countries. In Canada there is one vinyl chloride monomer factory (Dow Chemical, Fort Saskatchewan, Alberta), six PVC resin production factories and several major PVC resin compounding factories. Ontario has three PVC production plants, two (Dow and Royal Plastics) in Sarnia and the other (GEON Canada) in Niagara Falls, as well as several resin compounding factories. After many years of use, vinyl chloride has been recognized as a potent mutagen and carcinogen. Exposure to vinyl chloride has led to a large number of health effects and has been linked to a significant proportion of mortality among workers in the vinyl chloride and related industries. In this report we present an annotated bibliography of the literature relating to the genotoxicity and carcinogenicity of vinyl chloride. In addition, we provide many references on the pharmacology, etiology and potential health effects of vinyl chloride exposure as well as a brief overview of the information found within these references.

Acknowledgments

The authors wish to thank Dr. David Rokosh for his help in translating Dialog searches into more workable material. The authors wish to also acknowledge the assistance of Iraj Rahmani, who helped supply us with pertinent publications and Dr. Brendan Birmingham, who discussed certain aspects of this survey with us and who, along with Andrew Chiu and Adam Socha, kindly reviewed the manuscript.

101-02963/

1. INTRODUCTION

Vinyl chloride (CAS No. 75-01-4), an allylic halide-α-Cl, is a colourless, flammable gas at standard room temperature and pressure that has a mild, pleasing, ethereal odour. In its gas state vinyl chloride is approximately twice as heavy as air. When cooled vinyl chloride becomes liquid and it is in the liquid state that vinyl chloride is stored and transported. Vinyl chloride easily dissolves in most organic solvents but is less soluble in water (log K_{ow} 1.4-1.6). In the liquid state, under the correct conditions, vinyl chloride monomers will interact to form long chains resulting in the production of a polyvinyl resin. The resin is then modified by the addition of additives such as plasticizers. This process is referred to as 'compounding'. The compounded resin can then be used in the production of plastics, synthetic rubber, polyvinyl chloride (PVC) film, latex, PVC pipes, and many other compounds. The vinyl chloride monomer, which was first synthesized in 1833 (Binns, 1979), has been used in industry since 1931 when Germany developed a large vinyl chloride industry for the manufacturing of synthetic rubber. Most industrial countries of Eastern and Western Europe, Asia and North America have developed vinyl chloride industries. In Canada the vinyl chloride industry is concentrated in Alberta, Ontario and Quebec, though the Maritime provinces and British Columbia possess some of the secondary vinyl industry. Canada's first vinyl chloride factory was in Shawinigan Falls, Quebec, which began operation around 1941. Today, in Canada there is one vinyl chloride monomer production factory (Dow Chemical, Fort Saskatchewan, Alberta), six PVC resin production factories and several PVC resin compounding factories. Of the six resin factories, Quebec has one (Dow) in Varennes, Alberta is the site for two (Dow [Calgary] and GEON [Fort Saskatchewan]), and Ontario is home for the other three, two (Dow and Royal Plastics [previously owned by ESSO]) in Sarnia and the other (GEON Canada) in Niagara Falls. In addition, most of the major resin compounding factories are located in Ontario.

Through the years it has been noted that the production of vinyl chloride and its products has led to a number of health problems among factory workers as well as among people living in close proximity to the factories. The most serious of these, angiosarcoma, a fatal form of liver cancer, was discovered in 1974 by a B. F. Goodrich factory doctor who was able to associate vinyl chloride exposure with this very rare form of cancer. Subsequent studies have confirmed the link between angiosarcoma development and vinyl chloride exposure. In addition, some of these studies have identified other health problems to which workers in the plastics industry may also be at risk. In this report we present an annotated bibliography of vinyl chloride which identifies those references relevant to several vinyl chloride issues. The search of the literature for vinyl chloride publications was designed to emphasize the association of vinyl chloride with genotoxicity and carcinogenicity, and it covers the literature through January 1998, though a few manuscripts published after January are also included. In particular, the literature related to vinyl chloride as a mutagen and carcinogen, as well as to the mechanisms by which the reactive moieties of vinyl chloride are derived, are extensively covered. Similarly, references are provided to the epidemiological and cohort studies undertaken by different companies and countries that investigated the causes of the sickness and mortality among previous vinyl chloride workers. Furthermore, a brief overview of the information found within these references is presented.

The regulatory information available indicates that several countries, including Canada and the United States, have developed maximum exposure limits for vinyl chloride in air and water, although these appear to pertain predominantly to the work environment.

1.1 Reviews

A number of reviews have been prepared on various properties and effects of vinyl chloride. Many of the reviews deal with very specific subjects, although there are some excellent general reviews. One of the best reviews was published by the Ontario Ministry of Labour (MOL) in 1981. Although there have been some changes in the vinyl chloride industry in Ontario since 1981, this publication still offers interesting details on the production of vinyl chloride in Ontario. The MOL publication, in conjunction with Environment Canada's 1986 environmental status report on the vinyl chloride industry, provides a wealth of information on the history and status of vinyl chloride production in Ontario and Canada. The most current review on the mutagenicity of vinyl chloride is that of Giri (1995), while the most up-to-date toxicological review is the USEPA-IRIS publication(USEPA, 1988), which will be released shortly. A list of the reviews is provided below.

Anderson, M. W., D. G. Hoel, and N. L. Kaplan. 1980.
Apfeldorf, R., and P. F. Infante. 1980.
Badaracco, J. L. 1985.
Bartsch, H., J. Nair, and I. Velic. 1997.
Besemer, A. C. et al. 1984.
Blair, A. and N. Kazerouni. 1997.
Bogovski, P. 1980.
Chen, C. W., and J. N. Blancato. 1987.
Chu, K. C., and H. A. Milman. 1980.
Cole, P., and F. Merletti. 1980.
Corbett, T. H. 1977.
Dannaher, C. L., C. H. Tamburro, and L. T. Yam. 1981.
Doll, R. 1988.
ECETOC. 1988.
Epstein, S. S. 1975.
Falk, H. 1986.
Fishbein, L. 1976.
Fučić, A., D. Horvat, and B. Dimitrovic. 1991.
Giri, A. K. 1995
Grasso, P. 1976.
Hansen, E. S. 1990.
Hens, L., L. Verschaeve, and C. Susanne. 1977.
Higginson, J. 1976.
Hopkins, J. 1980a.
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IARC. 1986.

Infante, P. F. 1977.
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Karstadt, M. 1976.
Kraybill, H. F. 1976, 1981.
Lijinsky, W. 1979.
Lilis, R. 1981.
Loper, J. C. 1980.
Makarov, I. A., and I. V. Fedotova. 1983.
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Plugge, H., and S. Safe. 1977.
Poole, S., and N. A. Fenske. 1993.
Purchase, I. F. 1978.
Reif, A. E. 1981.
Shabad, L. M., and V. A. Genin. 1976.
Shevchenko, A. M., and A. P. Yavorovskii. 1981.
Sims, P. 1980.
Singer, B. A. 1988.
Sittig, M. 1981.
Taba, A. H. 1981.
Tsuchiya, K. 1991.
USEPA. 1980.
USEPA. 1998.
Uzych, L. 1988.
VomBruck, C. G., F. B. Rudolph, K. Figge and W.R. Eckert. 1979.
Wu, W. 1988.
Zaeva, G. N. 1976.

2. ROUTES OF EXPOSURE

Historically, exposure to vinyl chloride has been in the industrial workplace, in particular, from inhalation of factory air. As industry has made efforts to better protect workers from vinyl chloride exposure, attention has also been drawn to other avenues of exposure, such as environmental and commercial. A few papers address modified methods for detecting and monitoring for VC in air and water. Traditional methods involve adsorption onto activated carbon and desorption with carbon disulfide and analysis by gas chromatography using flame ionization for air and using an inert gas purge followed by gas chromatography or mass spectrometry for water samples.

2.1 Occupational - Human

Those individuals working in the production of vinyl chloride monomers or who use the VC resins in the manufacturing of PVC or PVC products, are at the greatest risk of vinyl chloride exposure. Within either industry the degree of exposure also varies depending upon the job. Although to a lesser degree, workers in associated industries, where PVC products are used in large quantities, may also be at some risk for significant exposure. These industries include the manufacturing of automobile parts, sportswear, pool liners, some home furnishings, the old vinyl record industry, et cetera. Similarly, a certain degree of risk, albeit smaller, exists in those industries where these products are assembled into items such as automobiles and toys.

2.2 Environmental - Human, plant and animal

Besides occupational exposure to vinyl chloride, humans can come into contact with vinyl chloride in the form of vapours being emitted from new car interiors, some furniture, and plastic materials, as well as from some food and beverages, and from air, soil and water sources outlined below. Plants and animals are also subject to exposure from these same air, soil and water sources.

2.2.1 Air - Outdoor, factory plume

Typically, the air in rural and urban areas of North America contain no detectable amounts of vinyl chloride (Stephens et al., 1986; Grimsrud and Rasmussen 1975; Hardov et al. 1984; Wallace et. al., 1984). However, the air near industrial sites at which PVC is being manufactured, poses some risk to residents within the natural dispersion from the factory's air stack. Studies of air samples in the proximity of an Ontario resin plant were conducted in 1995 and 1996 by the Ontario Ministry of the Environment (Ontario Ministry of the Environment, 1995, 1996). The studies measured vinyl chloride levels both upwind and downwind of the facility and sample locations extended out to two kilometers from the plant. No detectable levels of vinyl chloride were found in samples upwind of the factory but approximately 80% of the downwind samples exceeded the method detection limit (0.07 $\mu g/m^3$) of the analytical equipment. Of those samples exceeding the detection limit, 25% (1995) and 38% (1996) exceeded Ontario's current Point of Impingement interim standard of 3 $\mu g/m^3$ (Ontario Ministry of the Environment, 1995,1996). Effluents from large incinerators where PVC products are burnt may also contain vinyl chloride. When complete combustion is not attained, measurements of vinyl chloride in the air have ranged up to 105 $\mu g/m^3$ (Gordon and Meeks, 1977; Pellizzari et al., 1979, USEPA 1985).

2.2.2 Soil - (Landfill, leachate, sediment, soil gases)

Waste disposal and landfill sites containing material composed of vinyl chloride or PVC poses a potential for VC exposure. Vinyl chloride or associated products can leach into wells, surface water, sediment or they can be released in the form of soil gases. In addition, metabolic action by certain anaerobic bacteria of chlorinated aliphatics such as trichloroethylene, tetrachloroethylene and 1,1,1-

trichloroethane in soil or ground water may lead to a reductive dehalogenation of these compounds the will result in the generation of vinyl chloride (Smith and Dragun, 1984).

2.2.3 Water - Well, ground, surface, drinking

Water can be contaminated with VC from a number of sources, such as direct (liquid effluent or wastewater from factories) and indirect (air stack emissions) from PVC manufacturing plants, or from the leachate from waste disposal sites and landfills, as well as possible leachings from PVC pipes that carry water. Surface water would not readily retain vinyl chloride as water is not an optimal solvent for vinyl chloride (1.1 to 2.7 g/L at 25 °C, Cowfer and Magistro, 1983; USEPA 1985b) and therefore, should pass into the air column with time. Vinyl chloride can, however, leach into drinking water stored in certain types of plastic water bottles such as those used in Italy (Benfenati et al., 1991). The authors estimate an exposure level from these bottles that may exceed 100ng per person per day.

2.3 Commercial - Human/animal

The final products of the vinyl chloride industry, primarily polyvinyl wraps and bags are potential sources of human and animal exposure.

2.3.1 Food and Water

Once set, polyvinyl chloride by itself is a hard, brittle and inflexible material. To induce the flexibility needed for products such as food wraps, plasticizers are added. Polyvinyl chloride food wrappings, however, pose a potential problem as monomers and oligomers of vinyl chloride as well as some plasticizers can migrate into food from the wrappings. The migration of components, such as vinyl chloride, from plastics into food has been studied, both empirically and theoretically, by vom Bruck et al. (1978, 1979) and Bieber et al. (1984). Reports of the Working Party on Chemical Contaminants from Food Contact Materials (1989, 1993) indicate oligomers of vinyl chloride, though not dimers, were isolated from PVC wrapped food. Additional evidence that not all vinyl chloride oligomers migrate was supplied by Castle et al.(1996) who were unable to detect the migration of a vinyl chloride tetramer into food or a variety of beverages. It should be noted, however, that their limit of detection was 5-10 μg /kg.

As indicated above, vinyl chloride was also detected in drinking water from some plastic bottles (Benfenati et al., 1991). As well, the migration of vinyl chloride into drinking water from PVC pipe has been measured at levels ranging from 0.03 μg/L to 1.4 μg/L (Dressmand and McFarren, 1978). The wide range in measurements corresponded to the age of the system, with older pipe systems showing the lower vinyl chloride levels. To date, the levels of vinyl chloride detected in food and drinking water appear to be within the current acceptable ranges for food and drinking water.

2.3.2 Packaging and Containers

Besides food wraps and drinking water pipes and containers, there are other PVC products from which vinyl chloride and plasticizers can leach into substances used by man. These include some rigid containers as well as bags used in the storage or transport of food and medical solutions.

3. PHARMACOLOGY

The effect of vinyl chloride on the body is addressed in more than 100 references.

3.1 Etiology

The cause or origin of vinyl chloride-related diseases is associated with the degree of metabolism the vinyl chloride product undergoes within the system as well as the resultant metabolites that are formed and the final cellular targets of these metabolites.

Depending upon the metabolite that is generated, the target may be either a nucleic acid, or protein. The metabolites formed, all appear to be alkylating agents that will alkylate with DNA, RNA or proteins. Not all metabolites will bind to the same cellular targets; for example, chloroacetaldehyde, will bind to protein but not nucleic acids, while chloroethylene oxide, one of the more potent metabolites, binds exclusively to nucleic acids.

3.2 Pharmacodynamics

The action, effects and breakdown of vinyl chloride within the body have been investigated fairly extensively in rodent systems, as there are approximately 100 papers in which the formation and role of vinyl chloride metabolites are discussed. Vinyl chloride vapour is rapidly absorbed through the lungs and enters the blood. Within a few minutes the vinyl chloride can be detected in the liver, kidneys, bile and digestive system. If taken directly into the gastrointestinal system, vinyl chloride rapidly will move into the blood. Shortly after exposure, the greatest percentage of vinyl chloride within the system is found in the kidney; however, within a day or two the liver contains the largest percent of vinyl chloride and its metabolites.

3.2.1 Metabolism

The metabolism of vinyl chloride occurs primarily within the liver. In the liver, vinyl chloride is detoxified to polar groups which can be excreted in the urine once they have been conjugated to glutathione(Green and Hathaway, 1978, Jedrychowski, et al., 1985). Mixed function oxidases appear to play a major role in the metabolism of vinyl chloride, and the primary metabolite formed is chloroethylene oxide (CEO). As metabolism continues, a number of additional metabolites are formed from various metabolic pathways that are biologically significant.

3.2.2 Metabolites

There are several known vinyl chloride metabolites that appear to be capable of alkylating to cellular constituents. The list of metabolites includes chloroethylene oxide (CEO), chloroacetaldehyde (CAD), epsilon A, C and G, 2-chloroethane, chloroacetic acid, 3,N4-ethenoglutin, N2,3-ethenodeoxyguanosine, 1,N6-ethenoadenine, and 7-(2-oxoethyl)guanine. The role of each of these and other metabolites has been studied. The metabolites linked most often to the mutagenic and carcinogenic effects of vinyl chloride appear to be CEO and CAD.

4. HEALTH EFFECTS

A wide variety of deleterious health effects have been attributed to vinyl chloride exposure. However, prior to the 1974 discovery at the B. F. Goodrich plant in Kentucky, where the factory's doctor linked vinyl chloride exposure to angiosarcoma, most toxicologists believed that vinyl chloride did not pose a serious health risk. In fact, prior to 1974, the 'safe' exposure level was set at 200 ppm, rather than the five and under ppm levels which are standard for most jurisdictions today. The discovery in 1974 set in motion a series of events which led government, industry, labour and science to the realization of the hazards of vinyl chloride exposure. Subsequently, industry, labour and government all supported research into the biological consequences of vinyl chloride exposure.

4.1 General

Most exposure to vinyl chloride is through the air, either as contact with the skin or inhalation, and can lead to dermatological as well as internal health problems. Internal health effects are also experienced when vinyl chloride or its associated molecules enter the body via ingestion. Upon exposed to vinyl chloride in its liquid form an individual is susceptible to varying degrees of frostbite. As with almost all substances, the type and severity of any vinyl chloride induced effect is related to both the dose and duration of exposure.

4.1.1 Short-term

Limited exposure to vinyl chloride may induce a feeling of dullness, nausea, headache, eye irritation, neurotoxic effects, and dermatological effects. It is difficult to clearly define the duration of a short-term or limited exposure, and its effect will almost surely differ from person to person.

4.1.2 Long-term

There are many possible consequences of long term exposure to vinyl chloride. These include various forms of liver disease such as liver sarcoma and liver carcinoma, as well as diseases in other tissues such as lung cancer, digestive system cancers, cardiovascular disease, genetic defects and even melanoma. In the case of angiosarcoma, which appear to be the signature disease of long term vinyl

chloride exposure, the average length of exposure to vinyl chloride necessary to trigger angiosarcoma development has been estimated at 18 years, though a recent study has documented workers with as little as 3.5 and 4 years of exposure contracting angiosarcoma (Lee, et al., 1996). Another condition attributed to prolonged vinyl chloride exposure is a cold-induced pallor of the extremities, referred to as Raynaud's syndrome.

A potentially serious side effect of chronic exposure to vinyl chloride, even at low doses, is associated with the body's defense mechanisms. As noted previously, vinyl chloride metabolites are eliminated from the body after first conjugating with glutathione. Chronic exposure to vinyl chloride may significantly deplete the glutathione pool thereby reducing the defensive mechanisms of the body to subsequent electrophilic attacks by oxidative metabolites (Green and Hathaway, 1978).

4.2 Occupational diseases

Many of the health effects listed above are specific to workers in VC and PVC factories. These include sclero-dermiform lesions, acroosteolysis, Raynaud's syndrome and liver function abnormalities. Since cancer is the subject of most of the publications on occupational diseases, it has been set aside in a separate section below. This current section identifies the approximately 40 papers which address the non cancer occupational diseases relative to vinyl chloride exposure.

4.3 Mutation and Cancer

As indicated in the Introduction, the main thrust of this bibliography was the relationship between vinyl chloride and mutations and cancer. Therefore it is not surprising that there are more than 700 papers addressing these subjects.

4.3.1 Mutation

More than 300 papers cited here address the mutagenicity of vinyl chloride or related issues. Vinyl chloride has been shown to be mutagenic in almost all assay systems in which it has been tested. Phylogenetically, vinyl chloride has been shown to be mutagenic in all life forms tested including; bacteria, yeast, insects, plants, mammalian cells in culture, human cells, and whole animals. In fact, vinyl chloride is currently used as the positive control substance in many studies. Vinyl chloride is known to induce almost a full spectrum of mutations including several types of both point mutations and chromosome aberrations. Studies have indicated that certain chromosomes elicit a greater sensitivity to vinyl chloride than most of the chromosomes, suggesting some degree of specificity in the action of vinyl chloride (Fučić, et al., 1990b).

In the Ames Salmonella mutagenicity assay, vinyl chloride is mutagenic in the presence or absence of metabolic enzymes, although its mutagenic potential is significantly enhanced in the presence of mammalian metabolic enzymes. In particular, when either of the two primary metabolites of vinyl chloride, chloroacetaldehyde or chloroethylene oxide, is tested in the Ames assay, the mutagenic response is significantly increased over that for vinyl chloride tested in the absence of metabolic enzymes (Giri, 1995). Results with other mutagenic test systems support the hypothesis that vinyl

chloride is only a weak mutagen in the absence of metabolic activation. The greater risk is posed by the metabolic products of vinyl chloride.

4.3.2 Cancer

Some 400 references address aspects of the relationship between vinyl chloride and cancer. The monomer of vinyl chloride is a potent human carcinogen while only a weak to moderate animal carcinogen. As with many mutagens and carcinogens, vinyl chloride needs to be metabolized to an active moiety. Unlike many carcinogens, the carcinogenic metabolite(s) of vinyl chloride is considered a complete carcinogen; that is it does not need a co-carcinogen to produce cancer. In rodents, the mechanism by which vinyl chloride and its metabolites transform normal cells into the neoplastic state appears to initially follow the pathway involved in the development of genotoxic effects (Giri, 1995). The primary cancer produced by vinyl chloride is its signature disease, angiosarcoma. Sarcomas of the liver are extremely rare and have been strongly linked to vinyl chloride exposure. Evidence also exists, however, that links vinyl chloride to hepatocellular carcinoma, cancers of the digestive system, lung cancer, brain cancer and melanoma. Many cohort and epidemiology studies have attempted to correlate exposure to vinyl chloride with the above-mentioned cancers. In almost all cases, the clearest correlation is with angiosarcoma. Studies indicate that induction of angiosarcoma generally requires a fairly extensive exposure (~18 years) to vinyl chloride. Based upon at least one calculation, the latency period, the time from the initial exposure to the onset of the disease, for angiosarcoma is approximately 22.6 years. From this calculation, it has been estimated that by the year 2010 there will be another 500 cancer-related mortalities in North America and approximately 2000 more in Europe due to previous vinyl chloride exposure.

5.0 RISK ASSESSMENT

Allylic halides elicit approximately 100 times excess toxicity relative to neutral organic compounds with similar Log K_{ow} and molecular weights.

The issue of risk assessment as it relates to public health has become prominent in the last several years as vinyl chloride and other potentially genotoxic substances have been detected in soil gases and leachates from many landfill and waste disposal sites. Lifetime cancer risks have been projected from both rat oral and inhalation studies. Based upon information in the 1995 annual update of the US EPA's Health Effects Assessment Summary Tables, the increased cancer risk from exposure to 1 μg/L (assuming a 70-kg individual, drinking 2 litres per day) is 5.4×10^{-5}. This value corresponds to that used by the World Health Organization and the Ontario Ministry of the Environment. Based upon an Ontario Ministry of Labour guideline (1989), there is an increased cancer risk of one per million population associated with the inhalation of vinyl chloride at an ambient air level of 0.2 $\mu g/m^3$ and a risk of one per 100,000 with an exposure of 2 $\mu g/m^3$, these are time weighted averages. A similar inhalation risk value, 1×10^{-6} from exposure to 1 $\mu g/m^3$ of vinyl chloride, is being considered by the US EPA but has not been finalized..

5.1 Occupational Safety and Exposure Limits

Since the greatest degree of exposure to vinyl chloride is among factory workers, the issue of occupational safety is addressed in several publications. With the advent of the setting of 'standards', exposure limits have been established to provide a level of safety for workers in the vinyl chloride industry. Prior to the mid to late 1970's, when standards were developed for most countries, exposure to vinyl chloride was established at 200 ppm. Today stricter limits of exposure exist for all countries. These limits differ slightly among countries. Canada and the US have an exposure limit based upon a time-weighted average of 5ppm. Canadian VC or PVC factories have installed alarms that are attached to monitoring systems. When the level of VC in the air reaches a set point of 2 or 4 or 10 ppm, the alarm system is activated. The establishment of exposure limits suggests that low chronic exposure to vinyl chloride may not be a risk factor, although this is still undetermined.

5.2 Threshold Limits

Besides maximum air and water concentration limits, there are studies listed here that address potential threshold limits for vinyl chloride. One of the primary conclusions drawn from these studies is that diseases such as angiosarcoma are induced in those individuals that have been subjected to a fairly prolonged exposure period at fairly high doses. Whether there is a threshold limit for low level chronic exposure is uncertain.

5.3 Regulations and Legislation

In September of 1974, eight months after a correlation between angiosarcoma and vinyl chloride exposure was observed, the US EPA established a national emissions standard for vinyl chloride which came into effect two years later when published as part of the US Clean Air Act (October, 1976). Presently, vinyl chloride has yet to be added to the US EPA's Integrated Risk Information System (IRIS), which identifies substances as carcinogens as well as other human health hazards. According to an EPA spokesperson, however, this is merely an oversight and vinyl chloride should be added to IRIS by June 1998. Canada's first regulations came in 1979 when Health and Welfare Canada in Section 7 of the Canadian Clean Air Act established standards for the maximum permissible emission of vinyl chloride into air from stationary sources. There is some question as to whether the current legislation covers emissions from soil gas and landfill leacheates. Each of the provinces with vinyl chloride industries established their own regulatory standards for air and water, although for the most part provinces have adopted federal standards. The Ontario Ministry of the Environment has established its own ½-hour Point of Impingement interim standard for vinyl chloride of 3 $\mu g/m^3$. The Ministry has also established acceptable levels for several water sources including drinking water (2 $\mu g/m^3$). Emissions from individual process stations within a vinyl chloride or resin factory are also given individual standards. The Quebec and Alberta regulations mirror those of the federal regulations. Alberta, however, has regulations that also cover 'fugitive' emissions, which are emissions from as seal leaks, and other equipment failures. Regulation of fugitive emissions is the responsibility of the province.

6. RECOMMENDATIONS

Based upon the information in the search, it is clear that vinyl chloride and its effects on humans were studied extensively from 1974 to 1981. Most of the recently published material update epidemiological studies of vinyl chloride workers that were instigated in the mid-seventies. Today, with a half of a century of history and data to build upon, we have a better understanding of the actions and consequences of vinyl chloride exposure. This knowledge has led to the lowering of acceptable exposure values and the instigation of many safety practices. Unfortunately, more information is still required before the vinyl chloride picture can be completed. To help complete that picture the following actions are recommended:

- MOE and MOL should pay close attention over the next several years to the results of these follow up studies to help better judge the populations at risk as well as to better evaluate safe exposure levels, not only to workers, but also to communities situated in the shadow of heavy vinyl chloride industry.

- Efforts should be made to determine the risks involved from exposure to vinyl chloride that is emitted from waste disposal sites or landfills.

- More research should be channeled to determining the risks from low level chronic exposure to vinyl chloride.

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8. KEY WORD LIST

2-chloroacetaldehyde
2-dichloroethane
2-dichloroethylene
2-haloacetaldehydes
3-ethenodeoxyguanosine
3-ethenoguanine
Activation
Adducts (DNA, RNA and protein)
Air
Air pollution
Air pollution monitoring (See also monitoring)
Airborne pollutants
Alkylation (VC metabolites alkylate to DNA, RNA and protein)
Ambient
Ames Test
Angiosarcoma (Angio sarcoma or hemangiosarcoma)
Animal (Carcinogenicity and mutagenicity studies on animals, see also mice, rat and hamster)
Aquatic
Assessment (See risk assessment)
Atmospheric
Bacteria (As related to assay type used in mutagenicity studies)
Biodegradation
Biomonitoring
Bioremediation
Blood
Blood transfusion (VC migration from bag into blood)
Brain (As relating to disease and cancer)
Breast cancer
Canada
Cancer (See also angiosarcoma, brain, lung, digestive, and melanoma)
Carcinogenesis
Carcinogenic
Carcinogenicity
Carcinogens
Carcinoma
Case studies
Chloroacetaldehyde
Chloroethanes
Chloroethene
Chloroethylene
Chloroethylene oxide
Chlorooxirane
CHO (Chinese Hamster Ovary, See also hamster)
Chromosome
Chromosome aberrations

Cohort studies (Studies of individual factories worldwide)
Cytogenetics
Cytological
Cytotoxicity
Dechlorination
DEHA (di-2-ethyl hexyl adipate) a plasticizer
DEHP (diethylhexylphthalate) a plasticizer
Diagnosis
Digestive disease and cancer
Dimethylvinylchloride
DNA and VC metabolites
DNA adducts
DNA damage
DNA synthesis inhibition
Dominant lethal (mutagenic and toxic endpoint)
Dose-response
Doses
Drinking water
Drosophila melanogaster (mutagenic assay)
Early-life sensitivity
Escherichia coli (E. coli) (mutagenic assay)
Emission
Epidemiology
Epsilon G
Ethenoadenine
Ethenocytosine
Ethenodeoxyguanosine
Etiology
Exposure limits
Extrapolation (Bacteria and animal data to human)
Female (Risk of female workers and spouses to VC)
Fibrosis (liver disease and lung disease)
First association of vinyl chloride with angiosarcoma
Food (Contamination by VC and plasticizers from packaging)
Framentography (See also Methods of detection)
Gene
Gene mutation
General carcinogenicity or mutagenicity
Genetic
Genotoxicity
Germ cells
Ground water
Hamster (See also CHO)
Hazards
Health effects
Health records
Heart disease

Hepatic (See also liver)
Hepatic fibrosis (See also fibrosis)
Hepatocarcinogenesis
Hepatocellular carcinoma (See also carcinoma)
Hepatotoxicity
Histopathologic
Historical (Chronology of industrial diseases in some countries)
Host-mediated (mutagenic bioassay)
Human (Studies using human data)
in vitro (As pertains to mutagenicity studies)
in vivo (mutagenicity and carcinogenicity studies performed on whole organisms)
Indoor air pollution
Industrial
Industries
Industry
Inhalation
Kinetics (See also pharmacodynamics)
Landfill
Landfill gases (See also soil gas)
Latency
Leachate
Legislation (See also regulation)
Lethal
Liver (See also hepatic)
Liver cancer (see also angiosarcoma and hepatocellular carcinoma)
Liver disease (See also occupational disease)
Lung disease and lung cancer
Male (Epidemiological studies predominantly using men)
Malignant
Mammalian
Mammalian cells (As opposed to bacterial cell mutagenicity data)
Mechanism (VC metabolite interaction in mutations and cancer)
Melanoma
Metabolism (VC and its metabolites)
Metabolites
Methods
Methods of detection
Micronucleus
Microsome
Migration
Monitoring
Morbidity
Mortality
Mouse (Mice, mutagenic and cancer studies using mice rather than humans)
Mutagen
Mutagenesis
Mutagenicity

Mutation
N2,3-ethenoguanine
N4-ethenocytidine
N4-ethenocytosine
N4-ethenodeoxycytidine
N6-etheno-2'-deoxyadenosi
N6-ethenoadenine
N6-ethenoadenosine
N6-ethenodeoxyadenosine
Neoplasm
Neurospora (An organism used in mutagenic studies)
Neurotoxicity
Occupational
Occupational disease
Occupational health
Occupational safety
Olefins
Oncogenic
Ontario
Ordinance
Outdoor
p53 (most important protein in cancer research)
Packaging
Particulates
Patients
Pharmacokinetic
Pharmacology
Phytotoxic
Plant
Plasticizer (See also DEHA, DEHP, MEHP and plastisols)
Plastisols
Polish
Polychlorinated
Priorities
Promutagenic
Public health
PVC
PVC-processing
Rat (See also mouse, hamster: these identify mutagenicity and carcinogenicity studies using animal rather than human systems)
Raynaud's disease
Record of Decision
Regulations
Remedial
Reviews
Risk assessment
Saccharomyces

Salmonella typhimurium
SCE (Sister chromatid exchanges; a mutagenic endpoint)
Schizosaccharomyces(A yeast organism used in mutagenic studies)
Scleroderma
Scleroderma-like
Sister-chromatid exchanges (See SCE)
Skin (See also skin cancer)
Skin cancer (Also see melanoma)
Soil
Soil contamination
Soil gas
Spot test
Standards
Status of VC in industry
Statutes
Stillbirths
Styrene
Surface water
Surveillance
Swedish
TD50
Teratogens
Teratogenicity
Thorotrast
Threshold limits
Toxic
Toxic substances
Toxicological
Toxicology
Tradescantia
Transformation
Tumors
UDS (mutagenicity assay: unscheduled DNA synthesis)
Urine (mutagenicity analysis and urinary problems)
Vinylidene
Viral (See also phage and Lambda)
Volatile
Volatile organic compound
Water and water pollution
Well and well water
Women (See female)
Workers (Also work related matters)
Workplace
Yeast (In mutagenicity studies, see also Saccharomyces and Schizosaccharomyces)

9. KEY WORD INDEX

2-chloroacetaldehyde

Guengerich, F. P., J. R. Crawford, and P. G. Watanabe. 1979.

2-dichloroethane

McCann, J., V. Simmon, D. Streitwieser, and B. N. Ames. 1975.
Rannug, U., and B. Beije. 1979.
Rannug, U., and C. Ramel. 1978.
Rannug, U., A. Sundvall, and C. Ramel. 1978.

2-dichloroethylene

USEPA. 1990.

2-haloacetaldehydes

Guengerich, F. P., P. S. Mason, W. T. Stott, T. R. Fox, and P. G. Watanabe. 1981.

3-ethenodeoxyguanosine

Kusmierek, J. T., W. Folkman, and B. Singer. 1989.

3-ethenoguanine

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Activation

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Taylor, J. 1988.
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Air pollution

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Hauschild, N. T. 1991.
Holliger, C. 1995.
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